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Circular No. 556
July 1940 • Washington, D. C.
UNITED STATES DEPARTMENT OF AGRICULTURE

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A Flower-Spot Disease of Cultivated Azaleas

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INTRODUCTION

In April 1931 a hitherto unrecognized disease attacked the flowers of cultivated azaleas in gardens near Charleston, S. C. The outbreak was so sudden and destructive according to observers that hundreds of thousands of flowers at the height of bloom became discolored and softened overnight, and finally collapsed on the bushes. This occurred within 3 or 4 days from the first recognition of anything abnormal. Local residents, who were familiar with the blooming habits of azaleas in gardens where the disease occurred, were at a loss to account for the malady, as prior to its appearance there had been no departure from the ordinary cultural conditions and no adverse weather, such as frost or drought.

The same disease occurred again in February 1932, a season of exceptionally early azalea bloom. A survey of the azalea gardens in the vicinity of Charleston was then initiated by the United States Department of Agriculture, with the result that the new flower disease was found in all the estates visited within an area of 20 miles, extending from the city up both sides of the Ashley and Cooper Rivers.

In 1933 a systematic investigation of the disease was begun. Observations made revealed no relation between the age of plants, the stage of neglect or culture, or the amount or kind of fertilizer used. From a variety of fungi and bacteria isolated from infected flowers, one fungus of distinctive morphology was found so consistently as to

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suggest its causal relation to the disease. It was grown in pure culture and its pathogenicity to azalea flowers proved; but reproduction of the typical symptoms of the disease was not at first attained, because the fungus did not sporulate on artificial media and thus produce inoculum comparable to that occurring in nature. Consistent association of this fungus with the new flower spot in all localities where the disease has since appeared, together with repeated experimental inoculation and reisolation, have now established this organism as the specific pathogenic agent. Apparently neither the fungus nor the disease was mentioned in mycological and horticultural literature prior to 1933. Other fungi capable of infecting azalea flowers under some conditions also were found, but they showed none of the characteristic effects of the Charleston flower spot.

SYMPTOMS

Several stages in the development of flower spot may be recognized in the typically susceptible varieties of the Indian azalea group, such

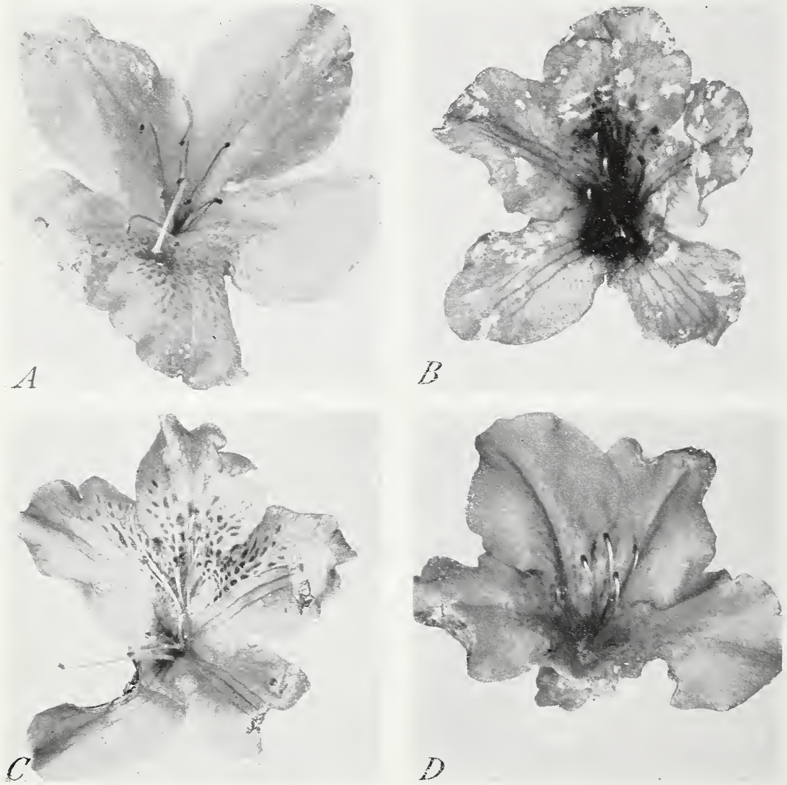

FIGURE 1.—Stages in the development of flower spot in lavender or pink azaleas: *A* (Variety Formosa), minute pale flecks, one, few, or many, on a single flower are the first visible signs of infection appearing 4 to 6 hours after spores reach the surface and germinate; *B* (Variety Duc de Rohan), the flecks enlarge and become blotches, with irregular margins; *C* (Variety "early lavender"), with further enlargement of the spots the tissues lose turgidity and the petals droop and shrink; *D* (Variety Formosa), one or more petals collapse.

as Formosa or Pride of Mobile. The first symptom is the appearance of from one to many small pallid spots, about 1 mm. in diameter, of circular form and smooth margin (fig. 1, *A*). They may occur on

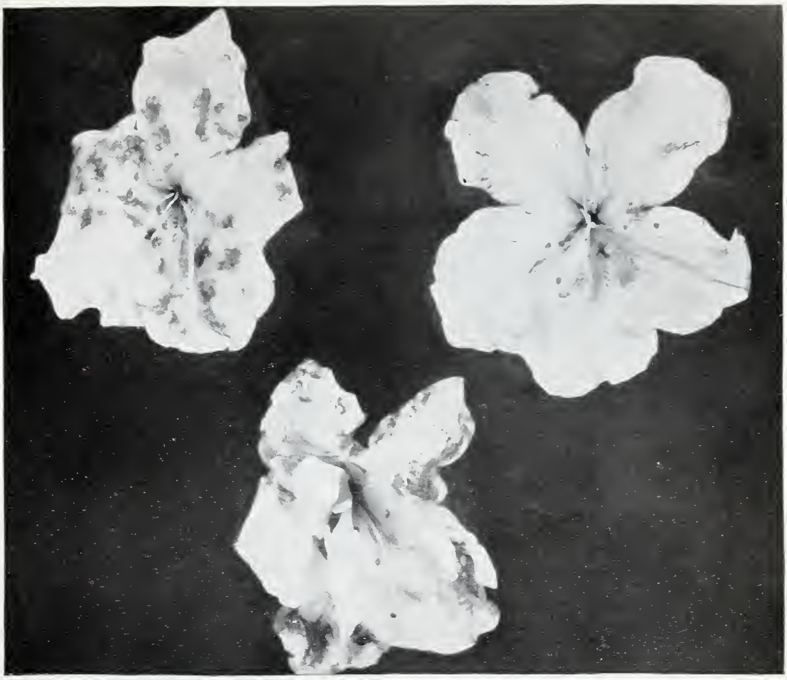

FIGURE 2.—Stages in development of flower spot in a white-flowered variety (Fielders White): The spots progress through the same forms as in colored flowers—flecks, irregular blotches, and limp blight—but they are rust-colored instead of white.

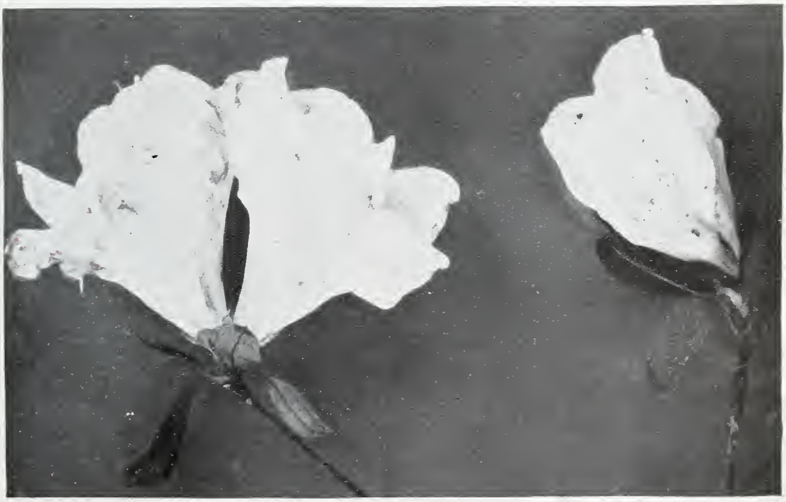

FIGURE 3.—Natural infection on outer surface of partly open flowers (variety Fielders White).

any part of the open flower but typically appear on the inner surface or face. In natural infections they are most numerous on the two lower and broader petals, and frequently they first appear at the sides of the opening into the corolla tube. Under favorable conditions they enlarge rapidly, reaching a diameter of 5 to 10 mm. in 24 hours (fig. 1, *C*). In rapidly enlarging lesions the margin becomes irregular with rootlike branching extensions, and the invaded tissue is soft and discolored. Copiously infected flowers have a freckled appearance when the spots are small, or blotched as the lesions enlarge and become irregular (fig. 1, *B*). The spots or blotches are brown or rust-colored on white flowers (figs. 2 and 3) and dirty white or gray on colored flowers (figs. 1 and 4). The loss or change of color due to disorganization and precipitation of the flower pigments can be readily seen (fig. 1, *C*).

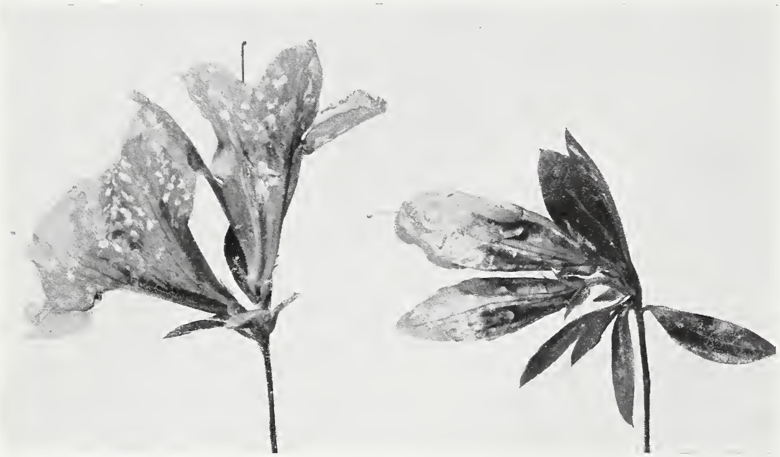

FIGURE 4.—Natural infection occurring on outer surface of partly open flowers and buds (variety Duc de Rohan).

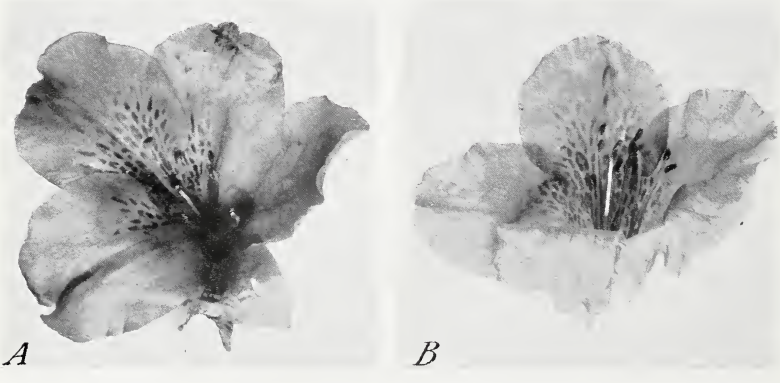

FIGURE 5.—Collapse of flower tissues (limp blight) without conspicuous spotting: *A*, Softening and slight discoloration of two petals; *B*, softening of entire flower (variety Formosa).

As the lesions enlarge further the tissue softens to the point of collapse, a stage appropriately designated as limp blight (figs. 5 and 6). Sometimes only a single petal collapses (fig. 5, *A*), but frequently the whole flower loses consistency and form, appearing to "melt" down and adhere like a wet blistered skin to the foliage (fig. 6). When all the flowers of a profusely blooming branch are simultaneously affected, a most unsightly effect is produced (fig. 7).

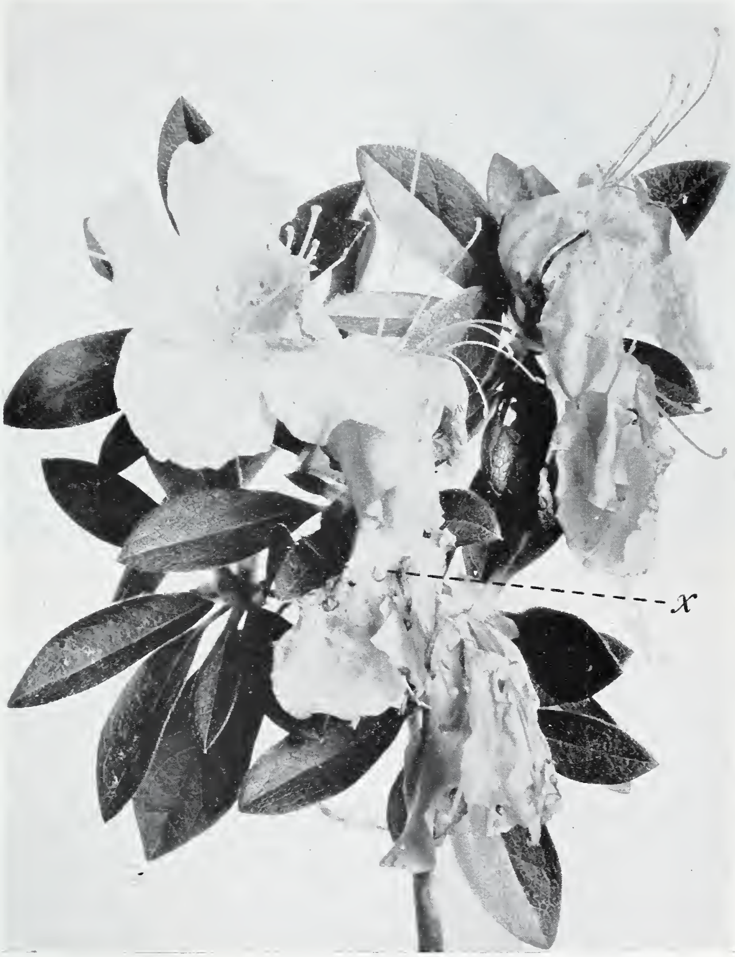

Figure 6.—Final stage of flower collapse. Resting bodies of the fungus (sclerotia) are beginning to form in the blighted flowers.

Ordinarily the course of the disease from the first visible spot to limp blight requires about 3 days, but the normal progress may be interrupted or delayed by dry, cold, or very hot weather. Sometimes only the small-spot stage is reached, or again the disease may be arrested in the large-spot stage without reaching limp blight. The flowers may then last for several days or a week, and the corollas then shed normally, whereas those that become limp-blighted usually fail to abscise and may stick so tightly to twigs and to foliage that removal by hand is required.

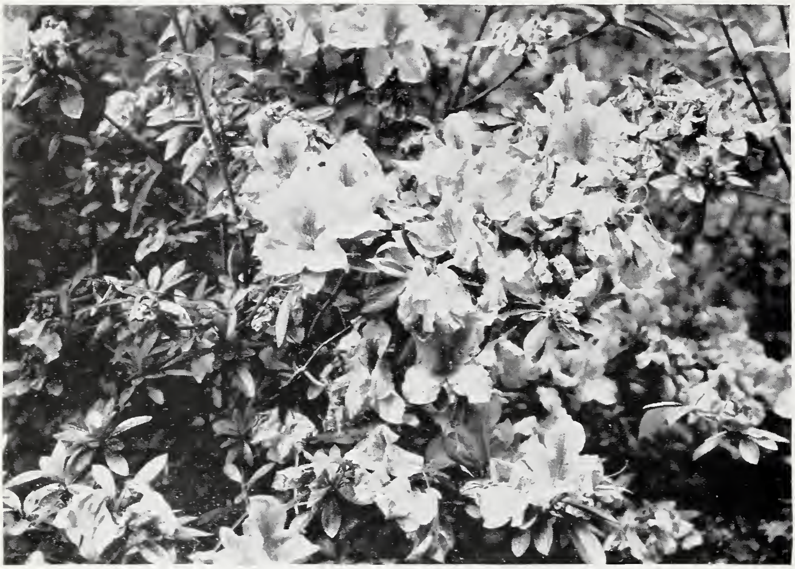

FIGURE 7.—Portion of azalea with most of the flowers blighted.

ETIOLOGY

The azalea flower spot is caused by a new fungus, which has been named *Ovulinia azaleae*.[2] The fungus is a new species and also the type of a new genus. It has many homologies with the group of discomycetous fungi that have been included in the old genus *Sclerotinia*, but that are now recognized as belonging to several genera distinguishable on the basis of the vegetative-growth forms, including the asexual spores, and of physiological and pathogenic behavior. The perfect stage of the azalea spot fungus is an apothecium typical of *Sclerotinia*, and it develops from a true sclerotium which overwinters on the ground or lightly covered with soil (fig. 8). The asexual spores, or conidia, which are produced only on infected flowers at a certain stage of development of the lesions, differ essentially from those of any of the genera heretofore associated with a perfect stage of the *Sclerotinia* type. A close physiological relationship is shown with the flower- and fruit-infecting species of *Sclerotinia*, such as those causing blossom blight and brown rot of pome and stone fruits, and especially those that infect the floral parts and young fruits of several species of ericaceous plants including *Vaccinium* and *Rhododendron*.

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[2] A technical description of the fungus has been published in Phytopathology 30: 236–244, 1940.

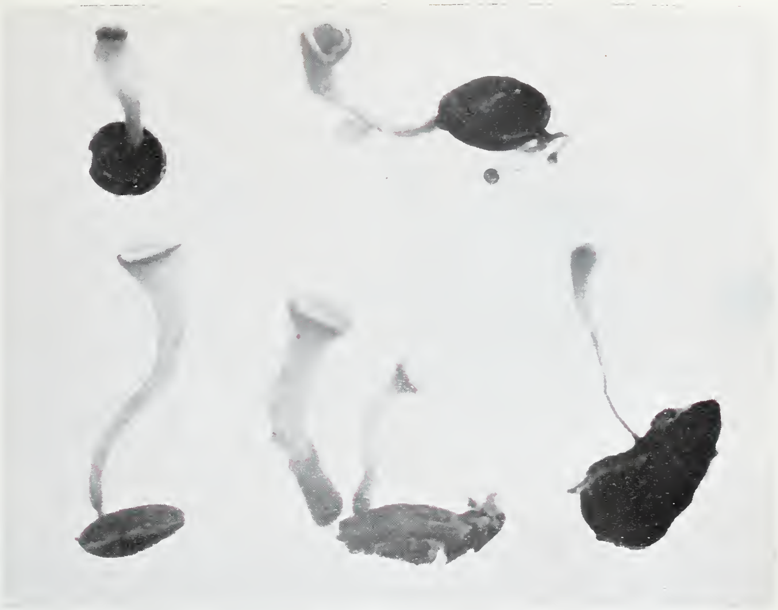

FIGURE 8.—Spring fruiting bodies or apothecia formed from sclerotia that have passed the winter on the ground or lightly buried. × 5.

LIFE HISTORY

The starting point in the seasonal cycle on azalea flowers is an apothecium, a minute fleshy fruit body of cup or plate form borne on a short stalk, which arises singly or in groups of two to three from an overwintering sclerotium (fig. 8) lying on or just beneath the surface of the ground. The cup portion at maturity is 1.5 to 2.5 mm. in diameter, and the stalk is 3 to 10 mm. long; the cup has a dull reddish-brown color, shading to light brown or buff in the stalk. The upper surface of the cup bears a palisade of closely packed spore sacs, or asci, each of which contains eight one-celled spores arranged in a linear series. At Charleston, S. C., the apothecia first appear coincident with the earliest sporadic bloom of the Indian azaleas, beginning about the middle of February to the first of March. The development of an apothecium from its first appearance to maturity takes about 3 to 5 days, and the period of ascospore discharge lasts 2 to 3 days more. The production of apothecia continues for some 4 to 5 weeks. The ascospores are discharged from the upper surface of the cup, which at maturity becomes flat, with sufficient force to reach low-hanging flowers, or they are caught by air currents and may reach flowers 5 or more feet above the ground. With suitable moisture the ascospores germinate and infect these flowers, producing primary lesions on which in a few days the secondary or asexual spores are borne. The secondary spores (conidia) are produced up to several hundred thousand on a single flower, and they serve to multiply the fungus very rapidly and cause infection of the successively appearing flowers. Not only are the secondary spores dispersed by air currents and raindrops, but they are carried by a number of flower-visiting insects, chiefly bees. As the cycle between successive crops of spores is only 3 to 4 days under optimum conditions, and with the profuse bloom characteristic of azaleas, enormous multiplication of infection is possible from a very small number of primary infections which are initiated from apothecia. It is not exceptional for the disease to spread to practically all the flowers of a large planting of azaleas, as of a nursery, park, or estate, or a community of small gardens, within 2 weeks after the initial occurrence of primary infections.

In limp-blighted flowers that persist on the bush, and in infected flowers that drop to the ground where they decompose under moist conditions, a final stage develops, which is of much importance in distinguishing flower spot from natural aging and from frost injury. This stage is the development of sclerotia or resting bodies of the fungus. They are black, disk- or cup-shaped bodies, 2 to 3 or sometimes up to 8 mm. broad, which are embedded in the petal tissue, usually in the thicker part, as the corolla throat or tube (fig. 9).

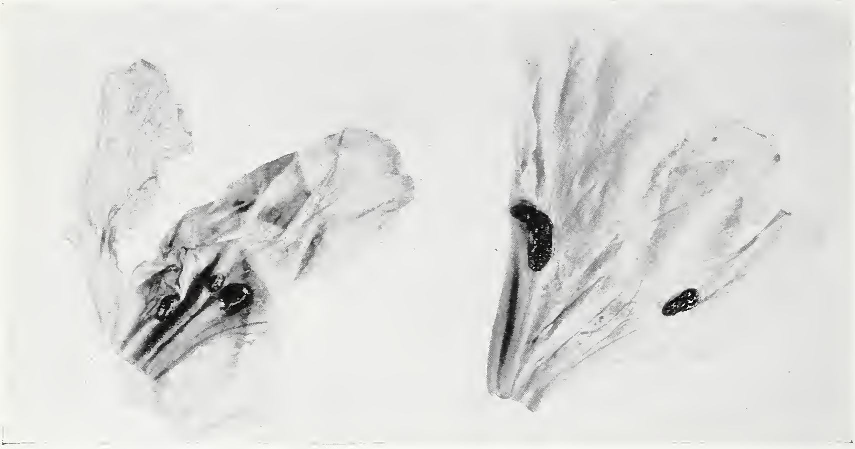

Figure 9.—Mature sclerotia produced in blighted flowers. Note pitted and moist surface characteristic of sclerotia when first formed.

Sclerotia are also produced in cultures of the fungus on artificial media. As the petal tissues of the fallen flowers disintegrate, the sclerotia are liberated and remain on the ground, or may be lightly covered by the washing and blowing of soil. Whether on the surface or slightly below, some of the sclerotia survive during the summer and ensuing winter, giving rise to apothecia again at the approach of the next period of azalea bloom. They may also carry through at least one flowering season in a dormant condition and produce apothecia the following year.

GEOGRAPHIC DISTRIBUTION

Although the azalea flower spot was shown by surveys to be rather generally distributed in the Charleston, S. C., district in 1933 and

1934, there were wide variations in its severity from place to place and from time to time during the period of azalea bloom. At times, when weather conditions were unsuitable for sporulation and infection, evidence of the disease nearly vanished; and certain gardens in the generally affected area showed such inconspicuous effects of the disease that only one acquainted with all stages of its development would have recognized it at all. In 1935 the aid of collaborators of the United States Plant Disease Survey and of nursery inspectors and plant quarantine officers in the States was solicited in determining how widely the disease occurred. Only a few specimens were submitted for identification in response to this request, and none was of this disease. Further explorations in the territory near the Charleston area disclosed the typical flower spot at Georgetown, S. C., and at Wilmington, N. C. Another local survey in 1936 revealed its presence at Savannah, Ga., and at Fairfax, S. C., and authentic specimens of the disease were submitted from Mobile, Ala., and New Orleans, La. In all cases the disease was found only on cultivated azaleas on estates, in home gardens, or in nurseries.

In 1937 an extensive survey was made in the Southeastern and Gulf States during February and March, and in the Middle Atlantic States from Virginia to Massachusetts during May. This survey showed that flower spot was present in most of the principal azalea plantings throughout the coastal area from Wilmington, N. C., to Avery Island and Lafayette, La., about 150 miles west of New Orleans, beyond which the survey did not extend. In the spring of 1938 the disease was found in park and home-garden plantings of azaleas at Jacksonville, Fla., and its occurrence at Palatka, Fla., was confirmed by the receipt of specimens after the period of azalea bloom, though it was not recognized there during the azalea flowering season. Flower spot has not been found in either outdoor or greenhouse plantings of azaleas north of Wilmington, N. C., and it has not been observed on native azaleas or other potential host plants belonging to the Ericaceae when growing under natural conditions, except in a few instances where they were in close proximity to infected plantings of cultivated azaleas. The disease was not found farther inland than Macon, Ga., and was not found at Jackson, Miss., or at Montgomery, Ala. However, negative findings in such a survey are of doubtful significance until repeated search has been made throughout the period of azalea and rhododendron bloom.

In 1939 the occurrence of flower spot on azaleas was reported from Houston, Tex. The disease was said to have been first noticed there in 1938 and to have seriously damaged the azalea bloom in 1939. This report has not been confirmed by the receipt of specimens.

Within this general zone there are local areas and sites where the disease has not been reported and may not yet be present. This is approximately the geographic range in which the Indian varieties of azaleas are successfully cultivated.

The source and sequence of the spread of this disease can only be conjectured. It first came to public attention at Charleston, S. C., but it is inconceivable that the severe outbreak of 1931 was its initial occurrence, as some time must have elapsed since its introduction during which it built up to destructive proportions. It has been repeatedly observed that even experienced nurserymen and gardeners may fail to recognize anything abnormal in the incipient and milder stages of flower spot, and even the more drastic effects may be misinterpreted, especially in the Kurume azaleas, being ascribed to natural aging or to frost. When a severe outbreak occurs with all the typical symptoms even the layman can hardly fail to recognize the condition as unnatural, but he is prone to regard it as of new and sudden origin. Making allowance for such errors of observation and deduction, it appears likely that the disease on cultivated azaleas originated at, or was introduced into, Charleston, S. C., at least as early as 1930, possibly several years earlier. It appeared at Savannah, Ga., and near Jacksonville, Fla., not long afterward, certainly by 1934; it was found at Mobile in 1936, and was reported to have appeared there the previous year. In Louisiana it was first observed in 1936 and in Texas apparently not until 1938. It may have occurred in these districts still earlier but escaped notice.

However, it is noteworthy that in Louisiana the disease occurs chiefly in plantings where young azaleas, of either the Kurume or Indian varieties, have been recently set. In some of the plantings of Indian azaleas, chiefly of the Formosa variety, which date back some 75 years and into which no recent introduction of azaleas has been made, the disease is conspicuously absent.

The disease is apparently unknown in Europe, according to statements made by correspondents in England, the Netherlands, and Belgium. The so-called Indian azaleas, which are actually natives of China, came to the United States via Europe where their culture and breeding have been carried on for many years without any flower disease having been observed.

No published record of flower spot has come from the Orient, the native home of numerous species and horticultural varieties of azaleas. Of the many types originating there, those that have chiefly influenced American azalea culture in the last 20 years are the species or groups of hybrids known as Kaempferi, Kurume, mollis, japonica, indica, and macrantha. Of these, only the Kurume and macrantha azaleas, which are evergreen, have become successfully established in the Southern States. The Asiatic deciduous types are not adapted to the hot summers of this region. The principal introductions of the Kurume hybrids and their rise to great popularity in American azalea culture occurred about 1920 and shortly thereafter; there is an approximate chronological coincidence between their introduction and earliest recognition of this disease.

HOSTS

Present information on the susceptibility of azaleas and related plants to flower-spot infection is shown in table 1. The group of hybrids known horticulturally as Indian azaleas, whose derivation is traced principally to the botanical species *Rhododendron simsii* Planch., *R. pulchrum* Sweet, and *R. mucronatum* G. Don, are generally susceptible. As the horticultural varieties available for observation and testing were predominantly of unknown and often complex constitution in respect to species, the probable reaction of these species to the flower-spot disease can only be inferred. Some of the oldest clones such as Indica alba and Phoenicia, generally accepted as approximating the species *R. mucronatum* and *R. pulchrum*, are susceptible to flower spot. No clonal variety equivalent to *R. simsii* was identified; therefore it can only be conjectured that this is a susceptible species, since of all the Indian azaleas observed, in the ancestry of which *R. simsii* is acceptedly involved, none showed a significant degree of resistance. The named varieties of Indian azaleas differ somewhat in the rapidity and extent of the development of lesions following infection, some showing conspicuous unsightly blotches within 1 day and collapsing in 2 or 3 days, others retaining their color and form for several days. The flowers of the widely popular varieties Formosa, Brilliant, Pride of Mobile, Compte de Nieuport, and Prince of Orange are prominently and quickly disfigured. The early-season varieties Vittata Fortunei, Elegans, and President Claeys often escape infection or show only moderate injury, because they usually flower before the seasonal rise of temperature has reached the optimum for the disease. The variety known as Indica alba or Ledifolia alba (*R. mucronatum*), one of the few relatively hardy Indian azaleas, is among the more tolerant kinds. The Indian azaleas, which are used largely for forcing and which consist of hybrids originated in Belgium and the Netherlands, such as Mme. Petrick, Professor Wolters, and Vervaeneana, are among the large-flowered types of soft texture that are conspicuously damaged. Varieties of the species known botanically as *R. indicum* (L.) Sweet (=*Azalea indica* L.) and horticulturally as macrantha azaleas are susceptible to experimental infection, but in the South they flower so late that they usually escape infection because the seasonal rise of temperature exceeds the optimum for the disease.

TABLE 1.—*Susceptibility of azaleas and related plants to flower spot*

[+ indicates positive infection, − indicates infection not yet observed but results not conclusive]

Horticultural group or species	Natural infection observed	Experimental infection obtained	Reproduction of fungus on infected flowers by—	
			Spores	Sclerotia
Indian azaleas (all observed varieties):				
Rhododendron mucronatum (Blume) G. Don	+	+	+	+
R. pulchrum Sweet				
R. simsii Planch				
Kurume azaleas (many varieties):				
R. obtusum (Lindl.) Planch	+	+	+	+
Other oriental azaleas:				
R. indicum (L.) Sweet	+	+	+	+
R. japonicum (A. Gray) Suringar	−	+	+	−
R. obtusum var. Kaempferi (Planch.) Wils	−	+	+	+
R. molle (Blume) G. Don	−	+	+	−
R. yedoense var. *poukhanense* (Lev.) Nakai	−	+	−	+
Native (U. S.) azaleas:				
R. arborescens (Pursh) Torr	−	+	+	−
R. austrinum (Small) Rehd	+	+	−	−
R. calendulaceum (Michx.) Torr	−	+	+	−
R. nudiflorum (L.) Torr	+	+	+	−
R. roseum (Loisel.) Rehd	−	+	−	−
R. vaseyi A. Gray	−	+	−	−
R. viscosum (L.) Torr	−	+	+	−
Kalmia latifolia L	+	+	+	?
R. carolinianum Rehd	−	+	+	−
R. catawbiense Michx	+	+	+	+
Gaylussacia baccata (Wang.) C. Koch	−	+	+	?
Vaccinium corymbosum L	−	+	−	−
V. fuscatum Ait	−	+	+	?
V. tenellum Ait	−	+	−	−

No attempt has been made to test the relative susceptibility of a large number of varieties in each horticultural group. Many varieties of the Kurume group have become heavily infected, and sometimes prominently disfigured, when growing in proximity to infected Indian azaleas. The pathogen has been observed to carry its life cycle through sporulation and sclerotium production on the flowers of Kurume azaleas that were infected naturally as well as experimentally. For the most part, however, the Kurume azaleas show less conspicuous effects of the disease, and the symptoms so closely resemble natural aging and frost injury (fig. 10) (which is frequent on these varieties in the South) that the casual observer would scarcely recognize them as diseased. Furthermore, the production of sclerotia in the flowers of this group is rare under natural conditions. In the variety Christmas Cheer, neither sporulation nor sclerotium production has been obtained in experimental inoculations.

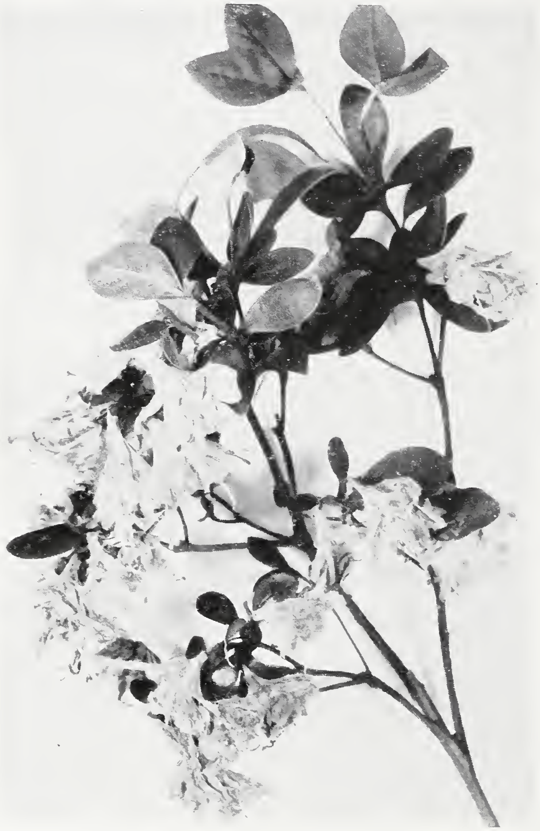

FIGURE 10.—Limp-blight stage of azalea flower spot on a Kurume variety (Pink Pearl).

Among other azaleas belonging to the Obtusum series of rhododendrons, the Torch azalea (*R. obtusum* var. *kaempferi*) and several hybrids derived from it (Atalanta, Carmen, Fedora, and Zampa) and the Korean azalea (*R. yedoense* var. *poukhanense*) have been experimentally infected with flower spot. Normal spore and sclerotium production ensued. The Obtusum series includes practically all the azaleas that are used extensively in greenhouse culture and many of the azaleas that are of commercial importance in outdoor gardening and in the nursery industry.

The native azaleas of the Eastern States are all included in Bower's canadense and luteum series of rhododendrons. Those of the southeastern Coastal Plain belong to the luteum series. The species *Rhododendron nudiflorum* and *R. austrinum* are sometimes included in ornamental plantings and are propagated to some extent in commercial nurseries. They (and also *R. atlanticum*) occur naturally within the range of outdoor culture of the Indian azaleas. All three species are susceptible to flower spot by artificial inoculation, and the first two have been found naturally infected when planted among or near (one-half mile) Indian azaleas, but limited observations thus far have disclosed no infection in isolated native plants. Normal sporulation has been observed on naturally infected flowers of *R. nudiflorum*. The native azaleas of the Piedmont, *R. calendulaceum* and *R. roseum*, have likewise proved susceptible to experimental infection, with the production of normal spores. In none of these species has the production of sclerotia been indubitably confirmed. The Piedmont species are not found in nurseries or in ornamental plantings within the range of Indian azalea culture, and they have not been found naturally infected. A thorough survey has not been made in their extensive natural range through the Piedmont and the Appalachian Mountains, but no flower diseases of these native species have been reported in mycological or pathological literature.

Of the remaining native azaleas of the Eastern States, the flowers of *Rhododendron arborescens*, *R. viscosum*, and *R. vaseyi* have been experimentally infected, but none of these species occurs within the present range of the flower-spot disease. The Pacific coast *R. occidentale* has not been available for testing. In general, it appears that the native azaleas of the United States are potential but not congenial hosts of the flower-spot fungus. Certain species might function in the dissemination of the fungus by the production of secondary spores, and possibly also in its perpetuation by sclerotia under exceptional conditions. The mollis-japonica hybrid series of azaleas appear to be congenial hosts when inoculated, but they have not been found naturally infected in the United States.

Among the potential hosts of azalea flower spot, two species of rhododendron in the horticultural sense and several other genera have been confirmed. Flowers of several unidentified hybrid rhododendrons, which were interplanted with Indian azaleas, became naturally infected with flower spot (fig. 11), and *Rhododendron catawbiense* and *R. carolinianum* have been experimentally infected with production of both spores and sclerotia. The mountain-laurel (*Kalmia latifolia*), when grown in proximity to infected Indian azaleas, readily contracts flower spot, and the life cycle is completed with the production of both spores and sclerotia. Finally the flowers of three species of *Vaccinium*, *V. corymbosum*, *V. fuscatum*, and *V. tenellum*, and of *Gaylussacia baccata* have been experimentally infected, with occasional production of spores and, doubtfully, of sclerotia, but no natural infections have been found. Attempts to infect flowers of other ericaceous plants, both native and cultivated, by inoculation with spores from azalea flowers have given negative or conflicting results, but the host range of the fungus cannot yet be regarded as completely known.

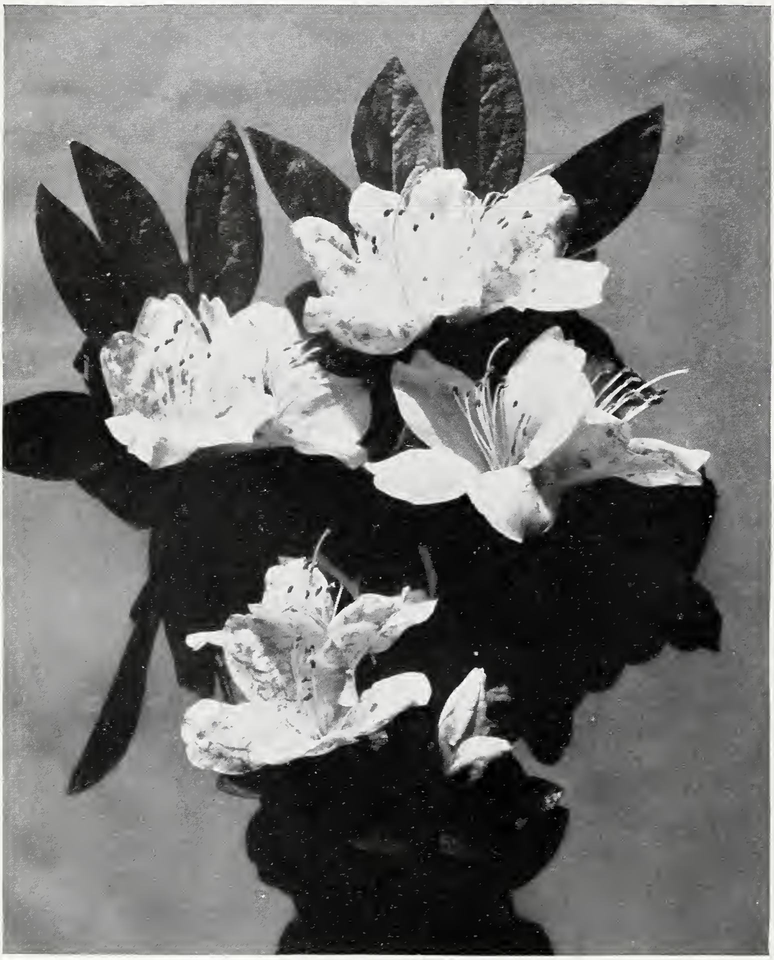

FIGURE 11.—Early stage of flower spot on rhododendron (catawbiense hybrid) flowers.

ENVIRONMENTAL RELATIONS

The spores borne on infected flowers are capable of germination within a temperature range of about 40° to 80° F., but normal infection of flowers is limited to a narrower range, which is approximately 50° to 72°. The optimum temperature, as determined from the proportion of successful infections and the rate of growth of lesions, is about 65°. Infection occurs readily without any mechanical injury, and is not facilitated thereby. The corolla is the only part of the flower, and the flower is the only part of the azalea plant that has proved susceptible to infection. Repeated inoculations on leaves, stems, and flower parts other than the corolla have all proved negative.

Although the conidia normally germinate promptly upon contact with water, when infected flowers collapse and persist on the shoots, the spores that are held within the folded petals may escape wetting and remain viable under natural conditions for several weeks. When such flowers were kept in dry storage at 40° to 50° F. the spores remained viable for fully a year. At higher temperatures, such as prevail during summer, they lose viability in 4 to 5 weeks. Although the spores that are retained by withered flowers or that lodge on foliage and within bud scales may remain viable for a time and thus afford a source of inoculum from which infection could occur or by which the fungus might be disseminated when such plants are transported, the viability of these spores is of relatively short duration. It seems very improbable that the conidia can perpetuate the fungus from one flowering season to the next. The effect of freezing temperatures on the spores and sclerotia has not been ascertained.

From a comparison of natural infection in the southern azalea gardens with the experimentally determined requirements for infection, it appears that the average seasonal conditions during the period of azalea bloom in the South afford about optimum conditions for the disease. The principal variable factor is the frequency and amount of precipitation, but in many of the typical garden sites in this area the frequent occurrence of dew, with slow evaporation under the canopy of Spanish-moss and evergreen foliage, provides the requisite conditions for infection even though rain is deficient or even lacking throughout the period of azalea bloom. The environmental conditions favorable to the disease do not differ essentially from the average conditions under which outdoor azaleas flower in the North and which occur in greenhouse culture, i. e., a temperature range periodically within 50° to 72° F. and occasional or frequent periods when the flowers are wet. The absence thus far of flower spot in greenhouse- or garden-grown azaleas in the North is probably to be ascribed to the nonintroduction of the pathogen, or to the occurrence of unfavorable conditions for its survival from one flower season to the next. Among these factors may be suggested the subfreezing temperatures that prevail in winter and the usually drier conditions of summer and fall, which in combination may be fatal to the sclerotia. In several instances flower spot has spread from test plants to a bench of stock azaleas kept in an experimental greenhouse that was managed according to ordinary commercial practice, and developed so rapidly that in absence of prompt control measures, all the azalea bloom in the house would have been quickly destroyed.

RELATIONSHIP OF INSECTS TO INJURY AND INFECTION OF FLOWERS

The rapid spread of the azalea flower-spot disease appeared not to be due entirely to the effect of wind or rain. Large numbers of insects observed to visit the azalea flowers were suspected of carrying spores from diseased to healthy blooms and thus of contributing to the rate of spread. Until the life history of the organism causing azalea flower spot, particularly the overwintering stages, had been completely worked out it seemed possible that insects were involved in the primary infection of azaleas each season, particularly in those infections occurring on flowers several feet above the ground. The entomological investigations were therefore planned to determine the relationship of insects to (1) the scratchlike abrasions in the corolla of flowers and the early-season scattered primary infections, (2) secondary spread of disease in a given planting and to widely scattered plantings, and (3) the introduction of the disease into the garden each year, either from some wild host or from overwintering quarters of the insect.

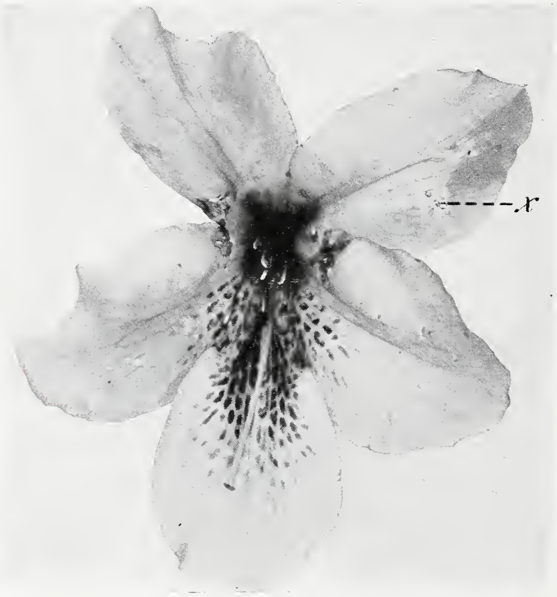

FIGURE 12.—Abrasions on lower left petal and opposite sides of the corolla deep in the throat of an azalea flower caused by spines on legs of visiting insects.

Insects have been very scarce on azalea flowers early in the season, but with the onset of warm weather and the approach of the period of full bloom, bumblebees (*Bombus* spp.), carpenter bees (*Xylocopa* spp.), and solitary bees (*Emphoropsis* spp.) visit azalea flowers in large numbers, together with a varying number of other bees, flies, and thrips. Representatives of all species as they became available were included in the experimental tests in the azalea gardens.

RELATION OF INSECT ABRASIONS TO FLOWER SPOT

The characteristic scratches occurring in the throats of azalea flowers (fig. 12) in gardens were supposedly caused by claws or spines on legs of visiting bees as they forced their way into the corolla for nectar. In experimental tests, representatives of four species of bees were allowed to scratch and bite flowers. When these flowers were kept under very moist conditions white margins developed along the scratches that were similar to those developing on flowers in the garden during rainy parts of the season. The scratches on flowers kept in less moist conditions developed clotted purple and papery margins resembling those most often found in the garden during warm dry periods of the flowering season. The tests fully demonstrated the ability of the insects to cause injuries similar to those observed on azalea flowers under natural conditions.

The first flower-spot infections of the season were frequently observed on flowers bearing typical insect abrasions, but in experimental transmission tests by insects over 92 percent of the flower-spot infections developed in the absence of visible mechanical injuries. The association of fungus infection and insect abrasions on the same flowers, often observed in nature early in the spring, is apparently only coincidental.

Disease-Transmission Tests With Insects

In the early investigations of the insect transmission of the flower-spot fungus it was definitely shown by microscopic examination that insects captured on diseased azalea flowers carried spores on their legs and heads. During a 4-week period that demarked the season of greatest bloom, an average of 20 percent of all insects tested caused infection. Among the insects (fig. 13) that were at times very abundant on azaleas, the two species of thrips (*Frankliniella tritici* Fitch and *Heterothrips azaleae* (Hood) caused very few infections. The four species of bumblebees (*Bombus americanorum* Fab., *B. bimaculatus* Cress., *B. impatiens* Cress., and *B. separatus* Cress.), the solitary bee (*Emphoropsis floridana* (Smith)), and the carpenter bees (*Xylocopa micans* Lep., and *X. virginica* Linn.) appeared to be most important in disease spread. Although honeybees (*Apis mellifera* L.) transmitted the disease in a few tests, they were apparently not important in disease spread.

In further extensive tests no infections were obtained with pollen taken from various species of bees on inoculated flowers even though the bees from which it was taken did cause infection.

Introduction of Disease Into Azalea Gardens Early in the Season

During 1937 and 1938 emphasis was placed on testing the possibility that insects were responsible for the earliest infections by bringing the organism from some early-flowering wild plant or from the over-wintering quarters of the vector and initiating infection in azalea gardens. In both years infections were observed among the earliest appearing flowers. During these early periods honeybees were practically the only flying insects present, and they were visiting camellia flowers. All honeybees and other insects captured on or near azaleas during the first 3 weeks of each season that the disease was present failed to cause infection. Large numbers of honeybees captured as they left the colony in a tree in 1937, or in two hives in 1938, caused no infection during the same periods. Many thousands of ants captured on azalea plants, or at bait traps beneath plants showing infec-

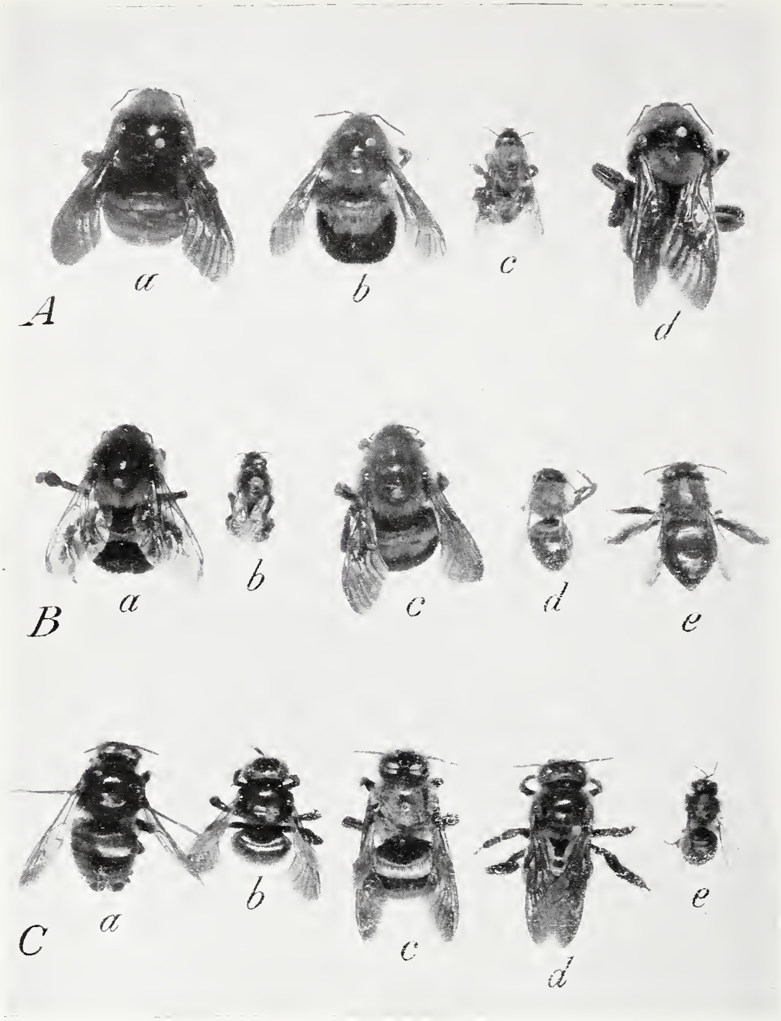

FIGURE 13.—Bees involved in transmission of azalea flower spot: *A*, *a*, *Bombus americanorum* (queen); *b*, *B. bimaculatus* (queen); *c*, *B. bimaculatus* (worker); *d*, *B. fraternus* (queen). *B*, *a*, *Bombus impatiens* (queen); *b*, *B. impatiens* (worker); *c*, *B. separatus* (queen); *d*, *Emphoropsis floridana* (male); *e*, *E. floridana* (female); *C*, *a*, *Xylocopa micans* (male); *b*, *X. micans* (female); *c*, *X. virginica* (male); *d*, *X. virginica* (female); *e*, *Apis mellifera* (worker).

tion the previous year, also failed to initiate infection. No infections were obtained with several types of insects, including ants, springtails, and flies, that were present in soil immediately beneath previously infected plants. The first positive infections by insects were obtained with bumblebees that appeared in the gardens about 3 weeks after the disease was present.

Occasional infections occurred in subsequent tests with bumblebees and carpenter bees, and, as the disease reached epidemic proportions, more insects became infective until over 90 percent of a given collection caused infection, and a single bee initiated as many as 107 infections. The results of the investigations indicate that insects were not responsible for bringing the disease into azalea gardens at the beginning of the season from their hives or overwintering quarters, from the soil beneath plants, or from other flowers, but that they became efficient carriers during the height of bloom at a time when infected flowers were already abundant.

Spread of Disease to Other Plantings and to Wild Hosts by Insects

The several species of bees that visit azalea flowers are extremely active and usually visit only a few flowers on one plant before moving to a nearby part of the garden, or even flying into the woodland or across the marshes. To obtain some information on the distance traveled by these insects, adults of bumblebees and of *Emphoropsis floridana* were marked with aluminum paint while visiting flowers in one garden. In 1 test in 1936, 3 adults of *E. floridana* and 2 of *Bombus impatiens* out of 150 marked adults of both species were captured on the same day in another garden 0.8 mile away. In a similar test, 1 adult of *B. bimaculatus* was captured in another garden 5 miles away 8 days after 150 adults of several species had been marked.

Bees that had been captured in two disease-free azalea nurseries located 0.4 and 1 mile, respectively, from any diseased azaleas were infective in experimental tests. These results, and others not related here, indicate that the insects move from one azalea planting to another for a distance of at least several miles and may distribute the flower-spot disease over the same area in a given season.

Bumblebees and carpenter bees of several species, and *Emphoropsis floridana*, have been observed to visit flowers of *Rhododendron nudiflorum* and *Vaccinium* spp. in the woods near infected azalea gardens. In one case flowers of *R. nudiflorum*, growing 0.5 mile from the nearest cultivated azaleas and showing typical feeding punctures of *Xylocopa virginica*, were found to be infected with flower spot. No natural infections have been found on flowers of *Vaccinium* spp., although both this wild host and *R. nudiflorum* have been experimentally infected with flower spot by insects.

The reason for the general absence of the disease on these wild hosts may be due in part to the appearance of bloom before or after the main flowering season of cultivated azaleas and to the onset of warm weather that is unfavorable for the development of the disease.

Control of Insects as a Means of Reducing Disease Spread

Since several species of insects (fig. 13) have shown themselves capable of transmitting azalea flower spot, the question arises as to the prospect of reducing spread of the disease by adequate control of the insect vectors. The insects involved are generally distributed throughout the Eastern States. Any control of these insects would therefore be limited to the locality near each affected azalea planting.

At present two lines of investigation on control of the insects seem possible. One is the provision of a succession of flowering plants extending over the normal flowering period of azalea that would be preferred to azalea by the insects. Azalea is not classed as a desirable honey plant by apiculturists. It is apparent in the present investigation that, with the possible exception of *Emphoropsis floridana*, all the bees and flies prefer certain flowers other than azalea. Honeybees feed in large numbers on camellias, holly, and *Photinia* while in flower and will avoid flowers of adjacent azaleas. The two species of carpenter bees (*Xylocopa micans* and *X. virginica*) were observed to feed on wisteria during its blooming period of nearly 3 weeks, after which they went to azalea flowers. Certain species of bumblebees appeared to prefer wisteria and white clover to azaleas when they had a choice. Further study of host preferences might reveal the possibility that a more complete succession of preferred host plants could be provided so that relatively few of the insect vectors would visit the azaleas and contribute to disease spread.

The second possibility is the direct control of the insect vectors by means of dusts, sprays, or poisoned baits. Standard contact dusts containing derris powder, pyrethrum powder, or nicotine sulfate had no apparent effect on bumblebees, carpenter bees, or *Emphoropsis*, whether applied directly to caged insects or to plants on which insects were later caged. Stomach poisons, as 15 percent of lead arsenate, 20 percent of sodium fluosilicate, or 10 percent of paris green, incorporated in the fungicidal dusts, also gave no evidence of control in cages. The most favorable results were obtained in cage tests with a sweetened bait composed of 5 percent of cane sirup in water to which were added as poisons either nicotine sulfate 1:200, derris powder to give a 0.0107-percent rotenone content, or tartar emetic 1:400. When these same materials were sprayed on flowers in the garden there was no evidence that any of these species of bees took any notice of the droplets of spray on the petals as they visited the flowers.

Certain observations made early in the investigation (p. 15) pointed strongly to the importance of insects as vectors of the flower-spot disease. However, as the potentialities of the organism were revealed by subsequent studies, the implication of insects as vectors was correspondingly reduced. After considering the present knowledge on the behavior of the azalea flower-spot disease no further investigations to perfect a direct control of the insect vectors are considered necessary. As previously discussed (p. 15), the disease organism is fully capable of initiating primary infection on the azaleas early in the season and of increasing rapidly until it destroys the majority of the flowers without the aid of insects. Although insects have been shown to be important vectors under certain conditions, their elimination will probably not materially affect the seasonal progress of the disease in a previously infected garden. Control measures against the disease itself are therefore essential. During the season of 1938 it was possible to compare the disease transmission by insects in two similarly situated, neighboring gardens; in one, no disease-control measures were carried out, with the result that the disease increased at the usual rapid rate; in the other, thorough and repeated applications of fungicide were made and infected flowers were systematically picked, which resulted in a marked retardation of disease spread. In the former garden infective insects were found in the first collection on March 1, whereas in the latter garden none were found that were infective until 2 weeks later.

In a further comparison of the disease transmission by insects in the two gardens, only one-third as many insects collected over the whole season were infective, and during the period of greatest infectivity the insects caused less than 5 percent of the number of flower infections in the garden where disease control was conducted.

The insects are doubtless involved in the distribution of the disease to local gardens. Because of the general distribution of the insect vectors and because of their varied life habits it appears that control measures directed against the disease will prove more feasible even under these conditions than will attempts to control the insects.

CONTROL

Since the phase of the life cycle of the pathogen that bridges the period from one azalea-flowering season to the next appears to be exclusively the sclerotial stage (figs. 8 and 9), the principal control efforts should be directed successively toward (1) the prevention of the formation of this stage, (2) the removal or destruction of such sclerotia as may form and reach the ground, and (3) the suppression of apothecial development by such sclerotia as otherwise escape. There might also be suggested for trial, especially in gardens containing large plants of which a few branches approach or touch the ground, that branches be removed or at least that their flower buds be picked, as numerous observations indicate that the earliest infections occur on flowers close to the ground. The first of these aims is accomplished by prompt and persistent picking of infected flowers as soon as the disease is detected. It may even be advisable to sacrifice the bloom entirely for 1 year and remove all the flower buds before they open, or at least as soon as any of them show infection. This procedure should be proposed only when the infected bush or planting is sufficiently isolated from other azaleas so that recurrence of infection from other sources is unlikely, or when the same procedure is carried out consistently throughout a community of plantings.

Except in plantings of relatively few and small azaleas, it will usually be found that the requisite degree of thoroughness in picking off spotted flowers so as to entirely prevent sclerotium formation cannot be attained, and some infected flowers will drop to the ground or sclerotia will form in those that adhere to the twigs or lodge in the branches. As azaleas are usually grown with a mulch of peat, leafmold, or leaf litter, the fallen flowers, and especially the sclerotia when freed from the flowers, cannot practicably be recovered. Complete removal of a mulch thus contaminated and its replacement with new material from a source known to be free from contamination is the next recourse to be suggested.

As an expedient in certain large azalea plantings where the complete renewal of the mulch was impracticable, a low level of apothecium production, as indicated by relatively sparse and late occurrence of the disease, was accomplished by applying a heavy mulch of rather loose dry leaves (recently fallen live-oak leaves are excellent) about 4 to 6 weeks after the azalea bloom was over. By this time practically all the flowers had dropped to the ground, and the thick coarse mulch was apparently an unfavorable medium for the production of fruiting apothecia or for the discharge of spores into the air. It has been observed that where azaleas are grown without a mulch (except for a temporary one during summer) or are mulched with fine-textured, well-decomposed leafmold, the disease is more prevalent than where a coarse, thick mulch of leaves is used.

In plantings where the disease has occurred for several years so that the soil is likely to be thoroughly contaminated with sclerotia, nothing short of extensive renovation of the surface soil, or turning it under deep enough to destroy or permanently inhibit the sclerotia, seems likely to be adequate. In nurseries it might be practicable to plow or spade the surface soil under in the space between plants. In gardens, although it would be impracticable to spade the surface soil under because of the broad and shallow root system of long-established azaleas, it might be expedient to scrape or brush up all the surface soil that can be removed without injury to roots, and replace it with at least a 2-inch cover of new soil or leafmold from an uncontaminated source.

It is not known how long the sclerotia may survive when buried in soil. The production of apothecia from sclerotia at least 2 years old has been observed, and sclerotia of normal internal and external appearance that were produced in an infected flower crop of at least 1 year previous can be found under azalea bushes when the current flowering season is well advanced but before a new generation of sclerotia has developed.

Some azalea growers have hopefully considered drenching the ground beneath the bushes with a fungicide as a means of either destroying the overwintering sclerotia or at least preventing the development of apothecia and the discharge of ascospores in the spring. No extensive experiments along this line have yet been made, but the possibility of thus exterminating the fungus is at best problematic. Because of their shallow roots, azaleas are likely to suffer chemical injury from any application potent enough to destroy buried sclerotia. Thus far all experiments making use of soil-surface applications of copper or sulfur fungicides commonly used as dormant sprays were ineffective in preventing primary infection. Possibly the timing of these applications was faulty, as they were not made to coincide with the period of apothecium development. Since this period covers at least 4 weeks, effectively timed applications would have to be repeated a number of times, increasing the risk of injury to azalea roots. At least one material of adequate fungicidal potency, and not appreciably toxic to azaleas even with applications repeated up to 10 times, is known. This is ordinary acetic acid diluted to give a concentration of acid between 0.10 and 0.16 percent (1 to 1,000 up to 1 to 600). Experiments with this and other fungicides, applied coincidently with evidence of apothecial activity and covering both the plant and the soil surface, are in progress.

As the fungus does not functionally survive over winter on the plants, or infect any part other than the more or less expanded flowers, it is evident that sprays applied only to the plant prior to the blooming period are futile. This has been amply confirmed by experiment. Blocks of azaleas in a woodland nursery, which were thoroughly and repeatedly sprayed up to and including the colored bud stage, showed not the slightest reduction or retardation of infection once the flowers opened and were exposed to either primary infection from ascospores, or to secondary infection from neighboring infected plants. Further evidence that the foliage and young flower buds do not bear the fungus, either superficially or internally prior to the widespread development of secondary infection, was obtained by cutting several hundred twigs with unopened buds and allowing them to flower in isolation from all other sources of infection, with the result that all the flowers remained healthy. After flower infection becomes established, however, buds and foliage may become contaminated with spores that give rise to flower infections even on twigs taken with only closed buds and flowered under cover.

The protection of open flowers by fungicidal sprays or dusts, though logically sound, is extremely difficult in practice. The waxy surface of the petals prevents ready wetting and it is practically impossible to attain thorough coverage of the dense masses of bloom that are characteristic of azaleas. Furthermore, spraying at very close intervals would be required as new flowers open every day for a period of several weeks. Both the flowers and foliage of azaleas are fairly tolerant of ordinary spray materials, and the pathogen is readily inhibited by concentrations of lime-sulfur and of bordeaux mixture or other copper fungicides that can be applied repeatedly to the flowers without evident injury. The difficulties in effective coverage are such, however, that all attempts have failed of complete control. However, these experiments have been conducted under conditions of such heavy and uncontrolled primary infection that the only valid conclusion deducible from the results is that control of secondary infection by spraying does not seem feasible.

It is much easier to obtain complete and uniform coverage of azalea flowers with dust than with liquid fungicides, and also to make applications in large plantings with the requisite frequency. Sulfur dusts are not sufficiently fungicidal, as the conidia germinate readily in drops of water containing sulfur particles and may infect flowers that are heavily coated with sulfur dust. In laboratory experiments, one application of copper-lime, or copper-clay dust, containing 6.5 percent of metallic copper, completely inhibited conidial germination, and infection of dusted flowers was reduced to less than 10 percent as compared with 100-percent infection in nondusted checks. Under outdoor conditions in a large garden, however, dusting failed to control secondary spread, even with applications repeated so as to produce a conspicuous and unsightly coating of dust on flowers and foliage. In one instance where primary infection was at a minimum because the planting had just been made on new soil (although the plants were taken from a contaminated nursery) the azaleas that received semiweekly applications of copper-clay dust showed only 1-percent infection during the flowering season, as compared with 5-percent infection in parallel blocks that were not dusted but were covered with cheesecloth to exclude insects. Comparable nondusted plants in the block from which these plants were moved showed nearly 60-percent infection at the same time.

It appears certain that overwintering sources must first be eliminated as far as practicable, and that if spraying or dusting is to be tried, applications must be begun as soon as any buds show color and before any open flowers are exposed to infection. Under these conditions, fungicidal applications made with sufficient thoroughness and frequency to reach all surfaces of the flowers and to keep new flowers covered as they expand may have some prospect of effecting control. Considering the ease of application, dusting with a copper-clay mixture of 5- to 6-percent equivalent metallic copper content is the most promising method, but it will be chiefly useful in nurseries where discoloration of the flowers and foliage is not particularly objectionable.

For ornamental plantings a colorless spray is required. For this purpose dilute acetic acid is worthy of further trial. A technical grade of acetic acid containing 80 percent of pure acid is cheap and readily obtainable through the larger drug-supply firms. It should be diluted 1 to 750 (approximately 1 pint to 90 gallons of water) for use on azalea flowers and foliage. This provides an approximate concentration of 1 part of acetic acid in 600 parts of water, or 0.166 percent. If acetic acid of some other initial strength is used (e. g., glacial or 100-percent acid) the dilution should be adjusted so that this percentage concentration is not exceeded, otherwise spotting of the flowers may occur. The 1-to-600 dilution may be applied freely to both flowers and leaves, and also used as a soil drench. As the acetic acid is completely soluble and is washed off by rain or dew, applications must be repeated after each period of precipitation. Spraying at intervals of 2 to 3 days will ordinarily suffice to restore the loss due to rain and to cover new flowers.

Acetic acid spray should not be used in spray pumps having porcelain cylinders, as it etches the porcelain surface.

From what has been said of the relation of flower-visiting insects to the spread of flower spot, it is evident that one azalea planting cannot be adequately protected if control is neglected in neighboring plantings. No definite limit can yet be set on the potential extent of dispersal of the fungus by insects, or even on the transport of viable spores by air currents alone, although evidence of spread by insects up to 3 miles has been noted. On the other hand, marked differences in the rate of development and the intensity of the disease have been observed in sites separated by only ½ to 1 mile, these differences being associated with the amount of primary infection and not with environmental factors. In communities where azaleas occur in almost continuous plantings nothing short of general cooperation will produce a high degree of control. It may even become necessary to establish some sort of community supervision to insure that neglect in one site may not undo all the efforts at control made elsewhere. The primary emphasis must be placed on the reduction of the number of surviving sclerotia to a minimum. If and when an effective dust or spray program is worked out, it may prove advisable in communities of extensive azalea plantings to establish a reporting service on the emergence of the apothecial stage of the fungus so that timely advice on when to begin spraying can be broadcast.

THE DISSEMINATION OF THE PATHOGEN ON NURSERY PLANTS

An important phase of control remains to be considered. This relates to the risk of local intensification of the disease, and also to its distant spread and introduction into new localities, by the transportation of infected or contaminated plants.

Several observations have been made demonstrating the introduction of flower spot into plantings previously known to be free of the disease by bringing in infected blooming azaleas from a nursery. In three instances the disease occurred again the following year without the further introduction of infected plants; that is, the fungus became established after one introduction. In another instance the introduced plants were uprooted and destroyed, though not until some local spread of the disease had occurred, which was promptly eliminated by picking off all exposed flowers. In this instance the disease did not recur.

Azaleas bearing infected flowers have been seen on sale in plant stores in Charleston, Mobile, and New Orleans. Usually the spotted flowers are not conspicuous or the worst ones are picked off by the dealers, and such plants are purchased without discrimination. Doubtless this practice has contributed greatly to the local spread of the disease. Together with natural dissemination of spores by air currents and by insects, the sale of infected plants may have already brought about so thorough a dispersal of the fungus in the centers of its present range that the suppression of such sales now would not materially affect its distribution. The situation is different in the border zone and in isolated areas of infection where there is grave risk of further spread by the transportation of infected plants. Even in the present centers of infection, such as the cities mentioned, obviously less progress can be made in control if there is no restraint on the sale and movement of infected plants. Nurserymen, plant dealers, and State quarantine officers should cooperate to eliminate this source of spread. Although recognizing that the most effective time for displaying and the preferred time for buying azaleas is when they are in bloom, and therefore the sale of blooming plants cannot be restricted without opposing current trade practices, growers and dealers should make it a rule to pick off all visibly infected flowers before the plants are sold. Purchasers of azaleas also should be careful to remove and burn all blemished flowers, and to pick the bush clean before the flowers drop off the first year.

The potential dispersal of the fungus upon or with plants is not limited to blooming azaleas. Sclerotia may be carried in the soil or in dead flowers that cling to the bush (fig. 14). Three instances of outbreaks of flower spot traceable to the presence of viable sclerotia in the soil that was moved with dormant plants have been observed. In one instance about 75 azaleas, 2 to 3 feet high, were lifted in January from a nursery where all the plants had been infected the previous 2 years, and without removing any of the surface litter from the root balls, they were transported by autotruck from South Carolina to Beltsville, Md They came into bloom about 3 weeks later and continued for a 6-week period. They were closely inspected daily. Three separate centers of infection, each involving 2 to 6 flowers on twigs close to the ground, were found on 2 of the 75 plants. The infected flowers were picked off before spores were produced on the lesions; secondary spread was thereby prevented, and following the removal of the primary infection there was no further development of the disease.

It should be emphasized that in commercial shipments the root balls of azaleas are normally cleaned of superficial leafmold and litter. As it is in this material that the sclerotia lodge, such cleaning should eliminate most of the risk of transporting sclerotia with the plants. Furthermore, a large majority of the commercial shipments of azaleas from nurseries in the Southern States to northern growers are of 1- or 2-year-old plants that have not bloomed and therefore have never been infected. Southern nurserymen could eliminate practically all risk of disseminating the flower-spot fungus on commercial shipments to northern forcers by isolating their azalea propagation as far as practicable from blooming plants, and by disbudding all plants during the flowering season preceding their sale. In the relatively local transportation of azaleas of flowering size, where disbudding may not be practicable, the risk of transporting the fungus in its sclerotial stage in the soil can doubtless be minimized by removing all the surface soil that can be spared from the top of the root balls before they are covered with burlap. Such plants should also be thoroughly cleaned of adherent flowers and of debris lodged between the crown shoots.

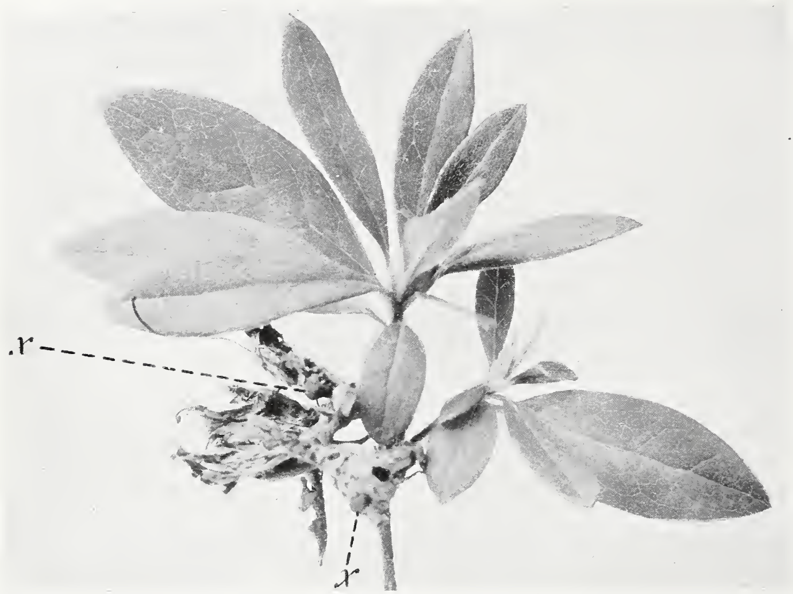

FIGURE 14.—Sclerotia in withered flowers that have persisted on the twig over winter.

As examples of the extent to which the risk of transporting the fungus can be reduced when these precautions are observed in transplanting azaleas, the following may be cited. In November 1935 about 3,000 plants 5 to 6 years old were moved from a nursery where flower spot had occurred for at least 2 years and were set in a city park. Not a single instance of the disease was detected on these plants the following spring. In December 1936 about 20 azaleas, 6 to 10 feet high, were moved from a bed where the disease had destroyed practically every flower during the preceding 3 years. Soil blocks 3 to 5 feet in diameter were cut out and balled after the careful removal of the surface litter. They were set in a site about 600 feet distant from other azaleas, and the root balls were covered about 6 inches deep with new earth and leaf mulch. Observations in 1937 showed that flower spot did not develop on these plants until they had been in bloom for 2 to 3 weeks and after the disease had become general on neighboring plants with secondary infection prevalent.

SUMMARY

A destructive disease, unknown prior to 1931, is described as attacking the flowers of cultivated azaleas, especially of the Indian and the Kurume types. It is caused by a hitherto undescribed fungus.

The disease causes spots on the flowers, which are first recognizable when about the size of a pinhead; they are pale or whitish on colored flowers and rust-colored on white flowers. The spots are initially circular in outline but enlarge rapidly into blotches of irregular form, and the affected tissue becomes soft and disorganized. Eventually the entire corolla collapses. Infected flowers tend to cling to the twigs, producing an extremely unsightly appearance. Under favorable conditions the disease may spread so rapidly as to destroy practically all the flowers of an extensive azalea planting within a few days.

The fungus produces resting bodies, or sclerotia, in the blighted flowers, which overwinter in the soil. From these sclerotia, fruiting bodies develop in the spring during the early part of the azalea flowering season; they bear spores which may infect only relatively few flowers, but secondary spores are produced in large numbers on the initial infections. The secondary spores are responsible for widespread and destructive outbreaks of flower spot.

Air currents and rain are the principal agents in the dispersal of spores from flower to flower or among closely planted bushes, but flower-visiting insects, especially bumblebees of several species, are important agents in the dissemination of spores to greater distances. The disease may be spread in this way to distances of 1 to several miles.

The fungus may be transported, in the secondary spore stage, on blooming azaleas and, in the sclerotial stage, in the surface litter and soil beneath dormant plants. When dormant plants are cleaned of surface litter and soil, as in the nursery practice of balling them for transplanting, dissemination of the fungus appears to be prevented.

The disease occurs in all the Southeastern and Gulf States from Wilmington, N. C., to Houston, Tex., and has been found in most of the prominent azalea plantings in the cities and towns in the Coastal Plain.

The first step in control is to reduce the survival of sclerotia to a minimum by destroying infected flowers before and after flower fall, and by removing the surface soil from around infected plants after flower fall and replacing it with new earth or a thick leaf mulch. Control of secondary spread by sprays or dusts applied to the flowers has not proved practicable without first reducing primary infections from overwintering sclerotia to the lowest possible minimum. Copper and acetic acid sprays and soil drenches, and copper-clay dust have shown promise of control under conditions of minimal primary infection and when their application is begun with the first appearance of colored buds.

U. S. GOVERNMENT PRINTING OFFICE: 1940

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